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Review

Tonsillitis and sudden childhood death

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Abstract

Critical reduction in upper airway diameter may result from tonsillar enlargement due to infection or from associated abscess formation. Other potentially lethal complications include hemorrhage and disseminated sepsis. Two cases are reported to illustrate features of specific cases: Case 1: a 12-year-old girl who exsanguinated from a pharyngocarotid fistula caused by a retropharyngeal abscess due to acute tonsillitis, and Case 2: a 17-year-old girl who asphyxiated from an aspirated blood clot following tonsillectomy. While most cases of acute tonsillitis resolve without sequelae, occasional cases may be associated with a lethal outcome. Massive hemorrhage may occur due to erosion of tonsillar vessels or subjacent larger vessels, or it may follow surgical extirpation of the tonsils. The autopsy assessment of cases where there has been possible lethal tonsillar pathology requires review of the presenting history and possible operative procedures, with careful dissection of Waldeyer's ring, adjacent soft tissues and major vessels. Presentations may not be straightforward and there may be misleading histories of epistaxis, hemoptysis, hematemesis and even melena.

Keywords: Tonsil; Adenoid; Hemorrhage; Asphyxia; Sepsis; Sudden death; Childhood

Introduction

Due to their critical position within the walls of the upper aerodigestive tract, the tonsils lie in proximity to major blood vessels and the oropharynx. This means that enlargement or infection may have a direct effect on airway calibre and vascular integrity. Although sudden deaths have been occasionally reported in the clinical literature there has been little forensic evaluation of the range of lesions and possible fatal mechanisms that may occur in cases of unexpected deaths associated with tonsillar pathology. For this reason the following study was undertaken.

Case report

Review of the pathology autopsy archives at The University of Adelaide revealed two pediatric cases where sudden death had resulted from tonsillar infection.

Case 1: A 12-year-old girl was admitted to hospital following two episodes of severe epistaxis. She was successfully treated for tonsillitis, although a swelling was subsequently noticed in the right posterior pharyngeal wall. She again complained of a sore throat with dysphagia and the swelling was noted to have increased in size. A third severe epistaxis controlled by packing was followed by massive hemorrhage and death.

At autopsy blood was dripping from her nose and a clot was adherent to the right posterior pharyngeal wall. Bilateral tonsillar enlargement was noted and dissection revealed a $2 \times 3 \times 4$ cm retropharyngeal hematoma with pseudoaneurysm formation around the right internal carotid artery. There was also adjacent cervical lymphadenopathy. Death was attributed to right internal carotid artery hemorrhage complicating tonsillitis with abscess formation.

Case 2: A 17-year-old girl underwent a right tonsillectomy with drainage for a right-sided post-tonsillar abscess. Two and a half hours after surgery she suffered cardiorespiratory arrest while using the toilet that was attributed to acute airway obstruction from a dislodged blood clot.

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Partial resuscitation was achieved, however death occurred several days later from hypoxic ischemic encephalopathy. At autopsy hypoxic encephalopathy was confirmed and the right tonsillectomy site was demonstrated covered with adherent blood clot.

Discussion

The tonsils consist of aggregated lymphoid tissue encircling the upper aerodigestive tract. The adenoids, or nasopharyngeal tonsils are found in the roof of the pharynx, the palatine tonsils lie on either side of the pharynx, and the lingual tonsils are situated at the base of the tongue. This mass of lymphoid tissue is known as Waldeyer's ring. The tonsillar tissues function as part of the immune system in dealing with inhaled and swallowed foreign antigens.

In childhood the tonsils may enlarge if they become infected with either bacterial or viral pathogens associated with acute pharyngitis. While most bacterial cases of acute tonsillitis in childhood are due to *Streptococcus pyogenes* other bacterial agents may be involved, as well as viruses that include Epstein-Barr and herpes.[1]

Generally the symptoms and signs of acute tonsillar infection are not serious and consist of a sore throat with fever, difficulty swallowing and signs of an upper respiratory infection. Resolution may occur spontaneously or with the assistance of antibiotic therapy. Rarely, however, tonsillar infection may result in serious and even life-threatening manifestations with a reported overall mortality rate of 1/1000–1/27,000.[2]

Lethal complications of acute tonsillitis include progression of sepsis and the development of airway obstruction. Septic complications may involve lethal dissemination of infecting organisms that have used the tonsil as a portal of entry. This was the case of a 9-month-old girl who was found unresponsive and who had a necrotizing tonsillitis due to *Clostridium perfringens* found at autopsy, with blood, cerebrospinal fluid and lung cultures positive for the organism.[3] Alternatively lethal septic complications may remain localized and lead to retropharyngeal abscesses that narrow airways, or to infections that extend deep into neck soft tissues and involve subjacent vessels.

Tonsillar infection may cause significant and spontaneous hemorrhage at any age including childhood, by causing (i) diffuse parenchymal hemorrhage, (ii) hemorrhage from small peripheral tonsillar vessels, or (iii) bleeding from major vessels due to erosion by deep abscesses.[4–7] Spontaneous hemorrhage is an unusual occurrence in children with acute tonsillitis, being documented in only 1.2% of cases in one series.[8] Although survival is possible with conservative management in cases of superficial hemorrhage, deeper infections that have involved major vessels may result in catastrophic bleeding with rapid demise, as in case 1 where there had been infiltration of the right internal carotid artery with pseudoaneurysm formation. While bacterial infections or infectious mononucleosis are usually associated with hemorrhage, on occasion bleeding in young adults has occurred in the apparent absence of either infection or trauma.[9,10] Younger individuals may present with hematemesis or melena from swallowed blood[11,12], or epistaxis as in the reported case, and episodic bleeding may be a marker for impending catastrophic hemorrhage.[13]

Hemorrhage may also complicate surgical removal of the tonsils but is more often found in elderly adult men than in children. Primary hemorrhage within the first 24 h of surgery, as in case 2, is more serious than secondary hemorrhage which usually occurs 5–10 days later.[14] Death may result either from simple exsanguination, or from hemoaspiration and obstruction of the swollen upper airway by blood clot, as in case 2. In children, it is the younger ages where there is a greater risk of secondary hemorrhage. The overall rate of postoperative bleeding in all age groups ranges from 1.5% to 4%[15–18] but may increase if an abscess tonsillectomy is performed. It also varies with the method of tonsillectomy used, with increased rates reported in some series where coblation (cold ablation) tonsillectomy was used.[2,19] A classification system for secondary hemorrhage has been proposed ranging from hemorrhage with spontaneous cessation (Grade 1) to hemorrhage with a fatal outcome (Grade 5).[20] The surgical treatment of significant hemorrhage may involve ligation of the external carotid artery, particularly if an aberrantly placed artery has been damaged during surgery.[21–23]

Airway obstruction in childhood may result from a variety of other conditions, ranging from foreign body inhalation to congenital cysts and tumors.[24–26] It may also be caused by simple hypertrophy of the tonsils and may be exacerbated by acute infection. Airway compromise usually occurs when there is bilateral enlargement of the tonsils, although this is not a prerequisite, as was observed in a 19-month-old boy who suffered acute airway obstruction when his glottis was occluded by an enlarged and pedunculated left palatine tonsil.[27] Problems with intubation due to lingual tonsillar hypertrophy have been reported in both children and young adults.[28] Hypertrophy of the palatine tonsils has also been hypothesized as a risk factor contributing to airway compromise in cases of sudden infant death syndrome (SIDS)[29,30], and children with adenotonsillar hypertrophy are known to be at increased risk for the development of obstructive sleep apnea.[31]

Tonsillar enlargement in conditions such as infectious mononucleosis may be associated with soft tissue swelling of the adjacent uvula and epiglottis, and/or peritonsillar abscess formation[32] and significant airway narrowing has been reported in 1–3.5% of individuals with this condition.[33] Tenacious mucoid secretions may also further compromise luminal integrity in such cases. In addition, sedation with narcotics may exacerbate underlying airway narrowing and was responsible for the death of a 14-years-old boy with recently diagnosed infectious mononucleosis who had significant tonsillar enlargement.[34] Although pseudomembrane formation is more characteris-

Table 1
Possible causes of sudden childhood death involving infection of the tonsils with autopsy findings

(1) Localized sepsis
(i) Airway obstruction with/without abscess
(ii) Airway obstruction with pseudomembrane formation
(iii) Hemorrhage with exsanguination and/or airway obstruction
(a) Parenchymal
(b) Tonsillar vessel
(c) Major vessel
(d) Postsurgical
(e) Idiopathic
(2) Generalized sepsis

tic of diphtheria, with membranes extending from the tonsils into the pharynx, larynx and tracheobronchial tree, pseudomembranes have also been described in infectious mononucleosis.[1,35] Dislodging of the pseudomembrane with airway obstruction may result in lethal asphyxia. Causes of sudden death due to tonsillar infections are summarized in Table 1.

The autopsy assessment of cases of unexpected and or sudden death associated with tonsillar pathology requires careful dissection of Waldeyer's ring, the tonsillar bed and the adjacent soft tissues of the neck, and the upper aerodigestive tract. This should reveal the nature and site of any lethal process and possible anatomical variants such as aberrant arteries that may have predisposed to a fatal outcome. However, prior to commencing dissection it is important to obtain a detailed history of the clinical presentation, including the chronology of medical intervention, and the type of surgical procedure used (if any), as complications may be related to operative methodology. Ancillary microbiology, virology and toxicology may also be required to clarify the contribution of various factors that may have led to the lethal outcome.

Conflict of Interest Statement

None declared.

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